## Reduced neural drive in bilateral exertions: A performance-limiting factor?

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# Reduced Neural Drive in Bilateral Exertions: A Performance-Limiting Factor?

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**ABSTRACT**

VAN DIEËN, J. H., F. OGITA, and A. DE HAAN. Reduced Neural Drive in Bilateral Exertions: A Performance-Limiting Factor? *Med. Sci. Sports Exerc.*, Vol. 35, No. 1, pp. 111–118, 2003. **Purpose:** Activity of the motor cortex in one hemisphere reduces the maximum motor outflow of homologous parts of the opposite hemisphere, causing a reduction in the maximum force a muscle can exert when the contralateral homologous muscle is activated concurrently. The purpose of this study was to establish whether this bilateral deficit is large enough to explain limitations in performance in bilateral exertions. **Methods:** Voluntary force production and neural drive during unilateral and bilateral exertions were compared in three experiments, consisting of unilateral maximum contractions, synchronous bilateral contractions, and asynchronous bilateral contractions of finger flexors and knee extensors. **Results:** Maximum voluntary force was overall about 7% lower in bilateral knee extension as compared with unilateral knee extension ($P < 0.001$). In finger flexion, a bilateral voluntary force deficit of as much as 20% was found ($P = 0.001$). Corresponding deficits in agonist EMG activity were also significant and on average found to be of similar size, though the magnitude of the bilateral deficit in EMG was not consistently related to the magnitude of the bilateral force deficit. In knee extension, a deficit in voluntary activation of 4% ($P = 0.003$) was demonstrated by means of superimposed tetanic stimulation. The magnitude of this deficit was correlated to the magnitude of the voluntary force deficit ($r = 0.80$, $P = 0.002$). The maximum rate of force development in bilateral knee extensions was 13% lower than in a unilateral knee extension ($P = 0.002$). **Conclusion:** These results suggest that deficits in bilateral force production are large enough to constitute an important performance-limiting factor. Furthermore, the data suggest that a reduced neural drive underlies this bilateral deficit. **Key Words:** BILATERAL DEFICIT, MAXIMUM VOLUNTARY CONTRACTION, MUSCLE FORCE, PERFORMANCE

Several studies have shown that activity of the motor cortex in one hemisphere reduces the maximum motor outflow of homologous parts of the opposite hemisphere, possibly through transcallosal inhibitory connections (7,16). The functional consequences of this inhibition have been subject of debate. It has been suggested that this inhibition is a factor limiting performance in bilateral exertions. Oda and Moritani (16) relate interhemispheric inhibition to a reduction in the maximum force a muscle can exert when the homologous muscle in the contralateral limb is activated concurrently. This reduction, commonly referred to as the bilateral deficit, has been demonstrated in several studies (3,6,17). It has been invoked to explain limitations in performance in bilateral exertions such as two-legged jumping (29). However, it has been questioned whether a bilateral deficit attributable to a reduced neural drive exists (10,11,13,29). Herbert and Gandevia (11) found no deficit in voluntary force and only a minute deficit in neural drive of the thumb abductor during bilateral contraction. Furthermore, they argue that evidence for a relationship between reduced neural drive based on EMG measurements and force deficit is inconsistent. They concluded that a bilateral deficit of substantial magnitude had generally been found only in large muscle groups. According to their interpretation, bilateral contraction of large muscle groups will lead to a problem in maintaining postural stability and consequently less efficient force transmission, thus causing a force deficit that is not dependent on reductions in neural drive.

In view of the disparate views in the literature, the aim of the present study was to further test the hypothesis that interhemispheric inhibition may result in a reduction of neural drive in bilateral exertions, as compared with unilateral exertions and in a concomitant reduction in maximum

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force. In a first experiment, the presence of a bilateral deficit was investigated in a small muscle group (finger flexors) to test the hypothesis set forward by Herbert and Gandevia (11).

Full activation of muscle in general appears to be difficult to achieve voluntarily (2) but can be achieved by highly motivated and well-trained subjects (4). The difficulty of achieving full neural drive causes a substantial variance in force output between attempted maximum voluntary contractions. This variance may obscure the bilateral deficit, because the deficit is determined by comparing separate unilateral and bilateral contractions. The study of Ferbert et al. (7) on interhemispheric inhibition, however, suggests an approach to study the bilateral deficit, which does not rely on repeated trials. These authors showed magnetic stimulation over the motor cortex of one hemisphere to reduce the excitability of the contralateral hemisphere by studying the electromyographic (EMG) responses to concurrent stimulation of this hemisphere. In addition, they showed that ongoing voluntary EMG amplitude of a muscle was reduced, when the motor area of the contralateral homologous muscle was stimulated. The latter suggests that a decrease in muscle activation and force output can be expected to occur also when during an ongoing contraction the contralateral muscle is voluntarily activated.

To further test the functional implications of the bilateral deficit and to verify its neural basis, a second experiment was performed on unilateral and bilateral knee extension. Neural drive was assessed by superimposed tetanic stimulation. This technique has been suggested to have a superior reliability as compared to superimposed twitch stimulation (9).

Finally, it has been suggested that fast dynamic contractions (1) and rapid force development (8) involve a stronger neural drive as compared with isometric contractions. This suggests that inhibitory effects of bilateral exertion on power output or rate of force development would be more pronounced as compared with the effects on voluntary isometric force. To test this hypothesis, a third experiment was performed with the same participants as experiment 2. In this experiment, the rate of force development was studied in unilateral and bilateral isometric knee extension, with the instruction to build up force at a maximum rate.

## METHODS

**Experiment 1.** Ten healthy right-handed subjects (five men and five women, aged 18–25 yr) participated in the experiment after signing an informed consent form. All experimental procedures were approved by the local ethical committee.

Each subject performed three attempted maximum isometric unilateral finger flexions of 5 s with each arm separately and three bilateral maximum contractions in a random order (protocol 1a). The subjects further performed three bilateral maximal contractions of 5 s in which the contraction of one arm started 1 s before the other arm followed. Five subjects started with right arm and 5 subjects started with the left arm (protocol 1b).

Finger flexion force was measured using a custom made dynamometer. Subjects were seated on a chair, the upper arms were vertical, and the elbows flexed 90° and fully supinated. The forearms were supported, and the wrist was in the neutral posture. The fingers were 90° flexed in the metacarpophalangeal joint and placed behind a horizontal bar instrumented with strain gauges. Force data were digitized at 1000 Hz. Throughout the experiment subjects were loudly encouraged.

Surface-EMG signals were recorded from the flexor digitorum superficialis and the extensor digitorum muscles, using bipolar disposable Ag-AgCl electrodes (Medi-Trace pellet electrodes ECE 1801, lead-off area 1.0 $cm^2$, interelectrode distance 2.5 cm). The signals were amplified (1000 times) and telemetrically transmitted to a Biomes-80 receiver (Glonner Electronic GmbH, Munich, Germany). EMG signals were band-pass filtered between 5 and 400 Hz before digitization at 1000 Hz. Finger flexion force was measured by flexing the fingers against a rigid bar instrumented with strain gauges. Force data were low-pass filtered at 50 Hz and digitized at 1000 Hz.

EMG signals were time shifted by 90 ms with respect to the force signals to account for the electromechanical delay. The maximum force (F) was determined as the average value over 500 ms around the peak force, and the maximum EMG amplitude was defined as the mean of the time-shifted rectified EMG of the agonist during the same 0.5-s period. The bilateral deficits for force and EMG in protocol 1a were determined as:

$$BLFa = 100 \cdot (F_{unilateral} - F_{bilateral})/F_{unilateral} \quad (1)$$

$$BLEa = 100 \cdot (E_{unilateral} - E_{bilateral})/E_{unilateral} \quad (2)$$

For the data of protocol 1b, the force (F) and rectified EMG (E) were averaged over 0.5 s preceding the onset of force in the second hand and over 0.5 s after force onset. The bilateral deficits for force and EMG were defined as:

$$BLFb = 100 \cdot (F_{before} - F_{after})/F_{before} \quad (3)$$

$$BLEb = 100 \cdot (E_{before} - E_{after})/E_{before} \quad (4)$$

The antagonist EMG amplitudes were determined over the same period as the agonist EMG amplitudes.

The trials in which the highest peak forces were obtained in each series of three repeated measurements were selected for further analysis. Repeated measures ANOVA were performed to test for differences in force, agonist EMG, and antagonist EMG between unilateral and bilateral exertions and between protocols (protocol 1a right arm, protocol 1a left arm, and protocol 1b).

**Experiment 2.** Twelve healthy subjects (six men and six women, aged 20–25 yr) participated in the experiment after signing an informed consent form. All experimental procedures were approved by the local ethical committee. The subjects visited the lab on two consecutive days. On the first day, they were familiarized with the procedures and trained to achieve near maximum activation as confirmed by

superimposed electrical stimulation (see below). The second day, all measurements were performed.

Each subject performed maximum isometric unilateral knee extensions of 3 s with the left leg and bilateral maximum contractions (protocol 2a). In addition, each subject performed maximal contractions starting with the left leg and after 1 s with the right leg (protocol 2b). Finally, subjects performed bilateral maximal knee extensions, relaxing the right leg after 1 s (protocol 2c). In all protocols, at least three successful attempts were made. Subjects were allowed to repeat any efforts they considered not to be maximal. Trials were performed in random order.

Isometric knee extension force was measured using a custom made dynamometer. Subjects were seated on a chair with a hip angle of 90 degrees. The pelvis and upper body were strapped with a belt and the lower legs were secured to strain gauges. Force data were digitized at 1000 Hz. Throughout the experiment subjects were loudly encouraged.

Surface-EMG signals were recorded from the vastus lateralis (VL) and the rectus femoris (RF) muscles, using bipolar disposable Ag-AgCl electrodes (Medi-Trace pellet electrodes ECE 1801 (Buffalo, NY), lead-off area 1.0 $cm^2$, interelectrode distance 2.5 cm). Signals were amplified 20 times (Porti-17$^{TM}$, TMS, Enschede, Netherlands), band-pass filtered (10–400 Hz) and A-D converted (22 bits) at 1000 Hz.

During protocol 2a, electrical stimulation was applied to assess neural drive of the left-knee extensor muscles. Stimulation was applied using a constant-current stimulator (Digitimer DS7H, Hertfordshire, UK) through self-adhesive electrodes (Schwa-Medico (Nieuw-Leusden, Netherlands), 80 × 130 mm). The upper electrode was placed over the quadriceps femoris muscle as far proximal as possible, and the lower electrode was placed such that its distal edge was 30 mm above the proximal edge of the patella. Before the actual measurements, four unilateral maximum contractions were performed. The current level of stimulation (with a frequency of 150 Hz) was set such that 50% of the maximum voluntary force was produced. Seven seconds before the voluntary contraction, a tetanic stimulation at the predetermined current, a frequency of 300 Hz, and a duration of 80 ms was applied (control tetanus); 1.5 s after initiation of the voluntary contraction, the same stimulus train was applied superimposed on the voluntary activity.

The maximum force (F) was determined as the average value over 250 ms around the peak force, and the maximum EMG amplitudes (E) were defined as the mean of the time-shifted rectified EMG of the RF and VL during the same 250-ms period. The two trials in which the highest peak forces were obtained in each series of repeated measurements were averaged to test for the presence of bilateral deficits in force and agonist EMG. The bilateral deficits for force and EMG in protocols 2a and 2b were determined according to Equations 1–4. For the data of protocol 2c, the force and rectified EMG were averaged over 250 ms after the decline of force in the right leg and over 250 ms preceding the decline of force onset. The bilateral deficits for force and EMG were defined as:

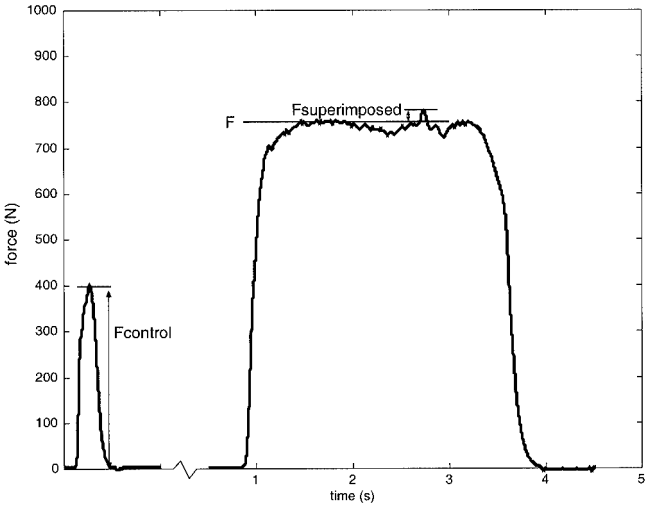


**FIGURE 1—Illustration of the measurements for the calculation of voluntary activation based on superimposed tetanic stimulation. Before the voluntary contraction a control tetanus is applied, the maximum force is defined as $F_{control}$. The voluntary maximum force is defined as F and the force elicited by the superimposed tetanus as $F_{superimposed}$.**

$$BLFc = 100 \cdot (F_{after} - F_{before})/F_{after} \quad (5)$$

$$BLEc = 100 \cdot (E_{after} - E_{before})/E_{after} \quad (6)$$

Neural drive (voluntary activation; VA) was estimated as:

$$VA = 100*(1 - F_{superimposed}/F_{control}) \quad (7)$$

with $F_{superimposed}$ being the peak force obtained during the superimposed tetanus minus the maximum voluntary force (F), and $F_{control}$ being the peak force during the control tetanus [Fig. 1, (9)].

Repeated measures ANOVA were performed to test for differences in force, VL and RF EMG, and VA between unilateral and bilateral exertions and between protocols.

**Experiment 3.** Experiment 3 was analogous to protocol 2a (in experiment 2), only the instruction differed. The same subjects participated again after signing an informed consent form. Subjects were instructed to increase knee extension force as fast as possible. The force signal was differentiated with a 5-point Lanczos filter, and the peak rate of force rise was determined as the average of a 20-ms period around the absolute peak rate. EMG data were full-wave rectified and low-pass filtered with a cut-off frequency of 25 Hz. Onset of EMG activity was detected from the data series using the algorithm provided by Staude and Wolf (25). The maximum EMG amplitude in the period of 250 ms after the onset was determined. The average over a 50-ms window around this absolute peak was used as an indicator of initial EMG activity.

## RESULTS

**Experiment 1.** A typical example of the forces produced in both protocols is shown in Figure 2. In protocol 1a,

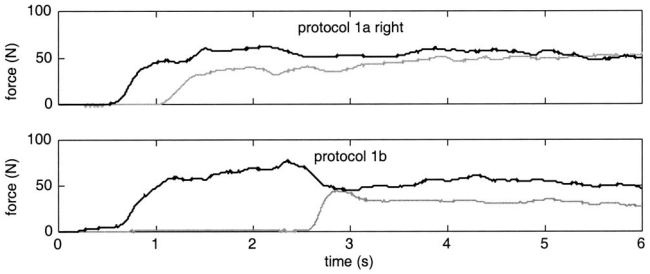


**FIGURE 2—Typical example of the forces in experiment 1. The finger flexion force of the right hand was higher in a unilateral (*black*) as compared with a bilateral contraction (*gray*) in protocol 1a (top window). In protocol 1b, the force of the right arm (*black*) that was activated first decreased after initiation of force in the left arm (*gray*; bottom window).**

the force in the bilateral contractions was in general lower than in the unilateral contractions (top window). In protocol 1b, the force produced with the arm that was first active declined at the onset of the force produced by the other arm (Fig. 2, bottom window). Note that the force decline appeared to start always close to the onset of force in the other arm well before the force in the other arm approached its maximum. Maximum voluntary force in bilateral finger flexion was significantly lower than in unilateral flexion (Fig. 3; $F_{1,9} = 22.19$, $P = 0.001$). For further analysis, for each subject data of protocol 1a were selected of the arm matching the arm first active in protocol 1b. ANOVA on this reduced data set revealed, as above, a significant difference between bilateral and unilateral exertion ($F_{1,9} = 25.6$, $P = 0.001$) and in addition a significant effect of protocol ($F_{1,9} = 10.8$, $P = 0.009$). Overall forces turned out to be lower in protocol 1b as compared with protocol 1a (Fig. 3, lower-right window) by 13% for unilateral contractions and 17% for bilateral contractions. No interaction effect was found, indicating equal deficits in the two protocols. However, the variance of the bilateral deficit was lower in protocol 1b (see Table 1).

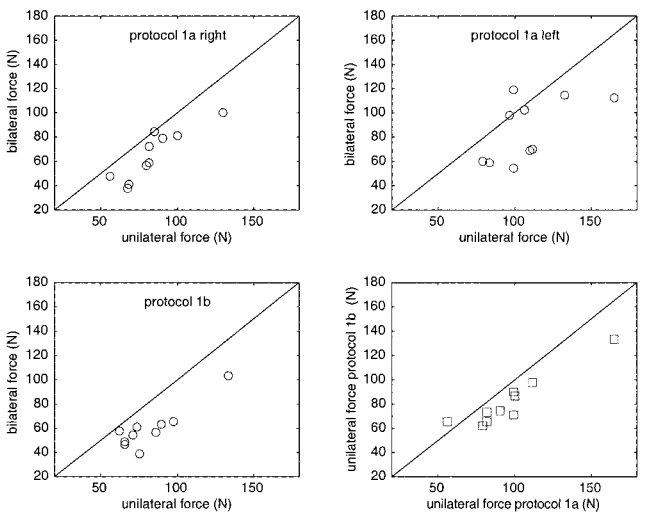


**FIGURE 3—Maximum finger flexion forces in bilateral and unilateral exertions in protocol 1a (top windows) and in protocol 1b (lower-left window). In the lower-right window, the unilateral force is compared between situations where there is no activity of the other arm (protocol 1a) and where the unilateral exertion is to be followed by an exertion of the other arm (protocol 1b). The *diagonal lines* are identity lines.**

TABLE 1. Bilateral deficits in finger flexion.

| | Mean | SD |
|---|---|---|
| BLFa left | 20.0% | 20.6% |
| BLFa right | 22.2% | 13.3% |
| BLFb | 26.9% | 10.7% |
| BLEa left | 19.7% | 22.1% |
| BLEa right | 20.3% | 25.1% |
| BLEb | 25.4% | 11.5% |

The effect of bilateral versus unilateral activity on the agonist EMG amplitude is illustrated in Figure 4. In protocol 1a, the agonist EMG amplitude in the bilateral contractions was in general lower than in the unilateral contractions (top window). In protocol 1b, the agonist EMG of the initially active hand was found to decrease immediately after the onset of force in the other arm in all subjects (Fig. 4, bottom window). In some cases, this suppression of EMG activity disappeared gradually. The agonist EMG amplitudes in the bilateral contractions were significantly lower than in the unilateral contractions ($F_{1,9} = 8.9$, $P = 0.015$). No difference between protocols was found, nor an interaction effect. There were no significant effects on antagonist EMG amplitudes.

The bilateral deficits in force were only moderately but significantly correlated to the deficits in EMG for the left arm in protocol 1a ($r = 0.75$, $P < 0.01$). The right-arm force and EMG deficits in protocol 1a were not significantly correlated ($r = 0.20$). In protocol 1b, again a moderate but significant correlation was found ($r = 0.64$, $P < 0.025$).

**Experiment 2.** Figure 5 gives a typical example of the force signals obtained in the three conditions. The results were qualitatively comparable to those of experiment 1; however, the bilateral deficit appeared smaller. The voluntary force produced in bilateral knee extension was lower than the force in unilateral extension (Fig. 6; $F_{1,11} = 41.0$, $P < 0.001$). Forces were lower in protocols 2b and 2c as compared with protocol 2a (Fig. 6, lower-right window; $F_{1,22} = 38.1$, $P < 0.001$) by on average 15% and 11% for forces produced unilaterally and by 20% and 14% for forces produced bilaterally. No interaction effect of protocol and bilateral versus unilateral exertion was found, which again

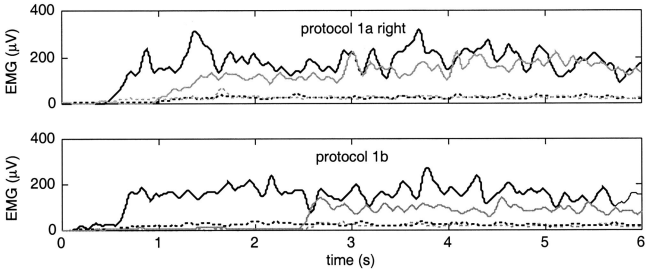


**FIGURE 4—Typical example of the EMG amplitudes in experiment 1. The EMG amplitude of the right hand was higher in a unilateral (*black*) as compared with a bilateral contraction (*gray*) in protocol 1a (top window). In protocol 1b, the EMG amplitude of the right-finger flexors (*black*) decreased after initiation of activity in the left-finger flexors (*gray*; bottom window). No effects were discernible on antagonist EMG amplitudes (*dashed lines, unilateral black, bilateral gray*).**

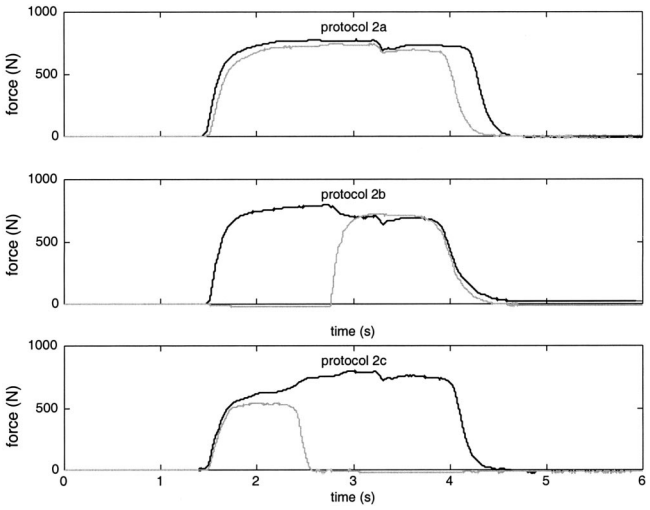


**FIGURE 5—Typical example of the knee extension forces in experiment 2. The force of the left leg was higher in a unilateral (*black*) as compared to a bilateral contraction (*gray*) in protocol 2a (top window). The force of the left-knee extensors (*black*) decreased after initiation of activity in the right extensors (*gray*) in protocol 2b (middle window) and increased (*black*) as the force in the right extensors (*gray*) started to go down in protocol 2c (lower window).**

indicates that none of the protocols showed significantly stronger deficits. However, as in finger flexion protocols 2a and 2c tended to yield higher deficits and clearly yielded a variance in deficits that was lower when expressed relative to the mean value (see Table 2).

EMG amplitudes of the VL were significantly lower in bilateral exertions ($F_{1,11} = 5.1$, $P = 0.045$). In addition, a significant effect of protocol was found ($F_{1,22} = 33.7$, $P < 0.001$), with amplitudes being 13–30% lower in protocols 2b and 2c. Similarly, the EMG amplitudes of the RF were significantly lower in bilateral exertions ($F_{1,11} = 19.8$, $P = 0.001$) and 6–26% higher in protocol 2a as compared with protocols 2b and 2c ($F_{1,22} = 13.9$, $P < 0.001$).

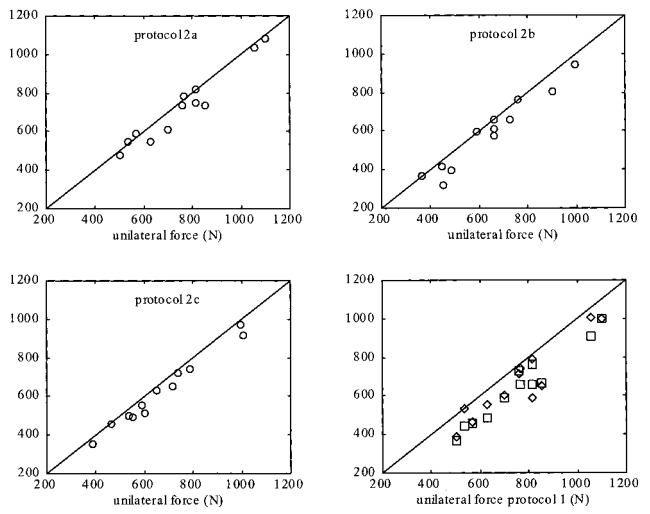


**FIGURE 6—Maximum knee extension forces in bilateral and unilateral exertions in protocol 2a (top-left window), in protocol 2b (top-right window), and protocol 2c (lower-left window). In the lower-right window the unilateral force is compared between situations where there is no activity in the other leg (protocol 2a) and where the unilateral exertion is to be followed or preceded by an exertion of the other leg (protocols 2b (*squares*) and 2c (*diamonds*)). The *diagonal lines* are identity lines.**

TABLE 2. Bilateral deficits in knee extension.

| | Mean | SD |
|---|---|---|
| BLFa | 3.5% | 5.6% |
| BLFb | 9.7% | 9.6% |
| BLFc | 6.9% | 3.9% |
| BLEa VL | 0.8% | 8.5% |
| BLEb VL | 5.0% | 22.1% |
| BLEc VL | 13.5% | 12.6% |
| BLEa RF | 0.5% | 15.3% |
| BLEb RF | 7.3% | 20.1% |
| BLEc RF | 18.3% | 9.8% |

The deficits in EMG were not significantly correlated to the deficit in force in protocol 2a (VL: r = 0.16; RF: r = 0.14). In protocol 2b, the correlations were significant for the EMG of both muscles (VL: r = 0.87, $P < 0.001$; RF: r = 0.84, $P < 0.001$). In protocol 2c, the results were inconsistent with a significant correlation for VL (r = 0.66, $P = 0.02$) and no correlation for RF (r = 0.09).

Voluntary activation (VA) in protocol 2a was on average 94% (SD 9%) in unilateral contractions and 89% (SD 9%) in bilateral contractions ($F = 15.0$, $P = 0.003$). The deficit in activation averaged 3.5% (SD 5.5%). The difference between VA in the unilateral and bilateral conditions was correlated to the bilateral deficit in voluntary force (Fig. 7; r = 0.80, $P = 0.002$).

**Experiment 3.** As is illustrated in Figure 8, the rate of force rise was generally higher in unilateral as compared to bilateral contractions. ANOVA revealed that the difference in rate of force development between unilateral contractions and bilateral contractions was significant (Fig. 9; $F_{1,11} = 17.1$, $P = 0.002$). The average deficit was 13 (SD 10%). No significant effects on EMG amplitudes were found. The EMG amplitudes averaged across subjects and unilateral and bilateral contractions were, for VL and RF respectively, 50% (SD 31%) and 31% (SD 30%) higher as compared to protocol 2a in experiment 2.

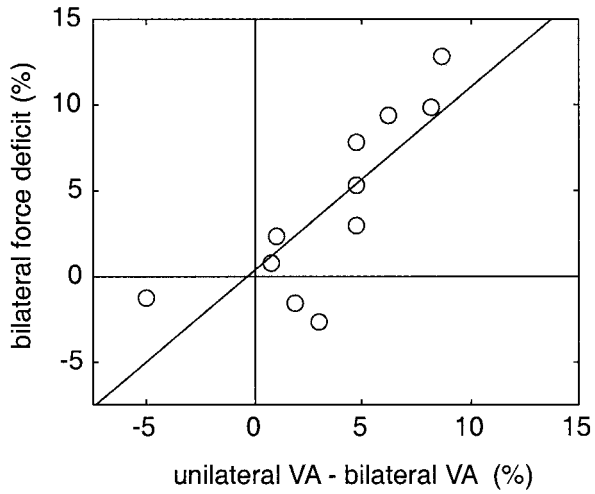


**FIGURE 7—The bilateral force deficit as a function of the difference in voluntary activation between unilateral and bilateral contractions. The *diagonal line* represents the regression line fitted to these data (r = 0.80, $P = 0.002$).**

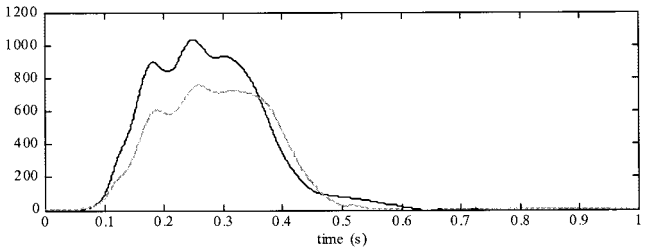


**FIGURE 8—Typical example of the force during a bilateral (*gray*) and unilateral (*black*) contraction of the knee extensors in experiment 3.**

## DISCUSSION

The existence of a bilateral deficit in force production was consistently confirmed in the present experiment. Furthermore, the data provide support for the assumption that this deficit was based on a reduced neural drive.

In both protocols of experiment 1, significant bilateral deficits in voluntary finger flexion force of about 20% were found. This average deficit is slightly higher than results obtained for the same muscle group by Ohtsuki (18). The range obtained in the study by Ohtsuki (−25 to + 44%) is also similar to that found when using protocol 1a in the present study. In experiment 2, the overall average bilateral deficit in voluntary knee extension force was 6.7%. This is within the wide range (0–18%) found for knee extensors in the literature (10,12–14,22,30). It should, however, be noted that in some of these studies no bilateral deficit was found (10,13), or it was found in a subgroup of subjects only (12).

In experiment 1, no effects of bilateral activity on antagonistic muscle activity were found. Therefore, the crossed extension reflex cannot account for the force deficit, as was also concluded by Ohtsuki (19). In all cases, deficits in agonist EMG activity were found, which were on average of approximately the same magnitude as the average force deficits. This appears to support the assumption that a reduced neural drive is the cause of the bilateral force deficit. However, a correlation between the deficits in force and in agonist EMG was not consistently found, and in most cases it was only moderate. In other studies, deficits in agonist EMG activity and moderate correlations to the deficits in force were found, in line with the present study (18,19). Some previous studies did show a deficit in force but not in EMG (12,22). In protocols 1a and 2a in the present study and in these previous studies, this may be a consequence of the variability between separate contractions, which is even higher for the EMG amplitude as compared with the force output. But also the within-trial variance of EMG amplitudes is more substantial than for force as can be seen in Figs. 2 and 3. In addition, for protocols 1b, 2b, and 2c, the fact that the time-shift between force and EMG was standardized across subjects, whereas a considerable between-subject variation in electromechanical delay has been shown to exist (5), might weaken the relationship between force and EMG deficits. Finally, the relationship between EMG amplitude and force often deviates from linear, especially at relatively high levels of activation (24). These limitations render EMG amplitudes fairly unreliable estimates of the level of voluntary activation. Therefore, experiment 2 comprised superimposed stimulation to estimate the level of activation as well. The VA was, with 94% and 89% in unilateral and bilateral exertion respectively, significantly different between conditions. These levels of VA are very similar to those reported by Jakobi and Cafarelli (13). In their study, this difference did not reach significance, however. The reduced VA in bilateral exertion found here provides strong evidence for a reduced neural drive underlying the bilateral force deficit. In addition, the relationship between the bilateral deficit in voluntary force and the difference in the level of voluntary activation was strong. Overall, these results suggest that a reduced voluntary neural drive of the agonist muscles is the cause of the bilateral deficit. The alternative explanation offered by Herbert and Gandevia (11), i.e., in bilateral exertions force transmission is less efficient due to problems in stabilizing posture, does not appear to hold for small muscle groups such as studied in experiment 1.

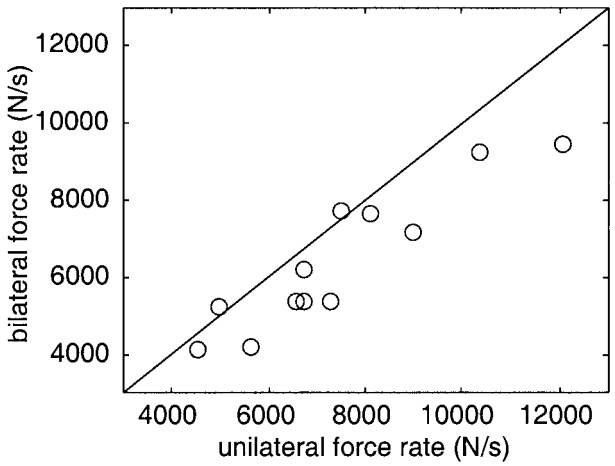


**FIGURE 9—Comparison of rate of force development during unilateral and bilateral knee extension. The *diagonal line* is the identity line.**

In experiment 3, it was shown that a bilateral deficit exists in the rate of force development. The deficit (13%) was lower as compared with earlier results obtained for the same muscle group by Koh et al. (14). Sahaly et al. (21) showed that when instruction to the subject combined emphasis on maximum force and maximum rate of force development, the deficit in rate of force development of leg extension were about 24%, whereas it was about 10% when only the rate of force development was emphasized. The latter result is in line with our findings. Because Koh et al. (14) stressed both force and rate of force development in their instruction, this could account for the differences found.

Several causes for the reduced neural drive in bilateral exertions have been suggested, (see for reviews 3,17). It does not seem likely that the cause would be a division of attention, because nonhomologous muscle activity does not result in a bilateral deficit (12). Although inhibition may occur at subcortical levels, studies of cortical activity suggest the presence of such interhemispheric inhibitory mechanisms (15), and transcallosal inhibitory pathways have been demonstrated between the primary motor cortices (7).

However, facilitatory pathways have also been demonstrated (28), and at submaximal force levels, consistent evidence of interhemispheric facilitation has been presented (26). This may in fact account for some of the disparate results in the literature. Furthermore, specific training can obviate the bilateral deficit in maximum force (20,23,27) and even result in bilateral facilitation (12). Thus, if interhemispheric inhibition is the cause of the bilateral deficit, this inhibition can apparently be overcome. The inhibition should probably be considered one of several plastic mechanisms inhibiting maximum neural drive, which explain the difficulty of achieving maximum activation of muscle in general. Not only the disappearance of the bilateral deficit with specific training but training effects in general have in part been ascribed to blocking of this inhibition (6). Direct evidence that training effects indeed operate at the level of interhemispheric inhibition is lacking. Blocking of inhibitory mechanisms by training might account in part for the difference in magnitude of deficits between experiment 1 and 2, as subjects were trained to perform the protocol only in experiment 2.

An unanticipated finding in the present study was the difference in the maximum voluntary force produced between the protocols in experiments 1 and 2. In the unilateral part of the asynchronous contractions (protocols 1b, 2b, and 2c), the force was lower as compared with the force in a simple unilateral contraction. Also the force during the bilateral phase of these asynchronous contractions was lower as compared with the force in a synchronous bilateral contraction. This was evidenced by the fact that a main effect of protocol was found, without any interaction with the activity being either uni- or bi-lateral. This suggests that the complexity of the instruction affected the level of force produced. Attention paid to the added task constraint might have caused a decreased attention for reaching maximum activation and thus account for this effect. It is interesting to note that this did not significantly affect the bilateral deficit.

The deficits in bilateral force found in the present study are clearly large enough to be functionally important and might for instance account for an important part of the 20% deficit in jumping height in two-legged jumps (i.e., 10% deficit per leg) (29). Because performance of such ballistic activities will depend not only on the maximum force but also on the maximum rate of force development, the relatively large deficit in this parameter underlines the relevance of inhibitory effects of bilateral activation of homologous muscles. Moreover, Vandervoort et al. (30) showed that the bilateral deficit strongly increases with movement velocity in concentric knee extensions to almost 50% at $424°\cdot s^{-1}$. However, the deficits in two-legged jumping described by van Soest et al. (29) were found in spite of the fact that the subjects were well-trained volleyball players, who can be expected to be specifically trained in two-legged jumping. Given the effects of training on the bilateral deficit discussed above, alternative explanations should also be considered.

In conclusion, the present study shows that in bilateral exertion neural drive can be reduced to such an extent that it will limit performance in maximum intensity activities. It is, however, clear from the literature that this bilateral deficit is not "hard-wired" and that it can be obviated by specific training.

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